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IRREGULARITY OF THE HEART AND AURICULAR FIBRILLATION.[1]

By ARTHUR R. CUSHNY, M.D., F.R.S.,
PROFESSOR OF PHARMACOLOGY IN THE UNIVERSITY OF LONDON, ENGLAND.

THE study of the pulse extends as far back as medical history, but it may be said without exaggeration that within the last fifteen years more progress has been made in certain directions than in the previous century. When the reason for this rapid elucidation of the subject is enquired into, it is found to be a simple one. Up to 1895, the human pulse only had been examined; since then the study has embraced the mammalian pulse; animal experiment has been called in to explain human abnormalities. Perhaps in no department of medicine has the employment of this new method of investigation—this *novum organum* of medicine—borne more immediate fruits. The whole of the new knowledge of the heart irregularities arose directly from experiments on animals. And I do not think I am doing injustice to the small band of investigators of this subject when I say that many equally talented and original minds had previously studied the irregularities of the heart, and that the recent success is due not to any preëminent ability on their part, but to the means of investigation they employed and to the application of the knowledge acquired in the laboratory to the problems of the sickbed. Another point of interest in this connection is that none of these investigators, so far as I know, had in his mind the clinical problem when he commenced his work. As has so often been the case, casual observations pursued merely for the sake of knowledge, or as our critics say, "out of idle curiosity," have led to the elucidation of clinical problems of the greatest importance. It is well to put on definite record that Gaskell and Englemann took up the study of the frog's heart without knowledge of its bearing on the irregularity of the heart in man, that my work on the mammalian heart was undertaken to elucidate irregularities seen in the dog's heart under poisonous doses of digitalis, and therefore without bearing on therapeutics, and that Hering's subsequent experiments were inspired by similar "idle curiosity," in regard to certain irregularities observed on tying the aorta. I think it well to make these remarks as the memory of the medical profession is short. One uses the knowledge or the remedies with thankfulness and neglects to enquire how they were obtained.

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[1] The inaugural Weir Mitchell Lecture of the College of Physicians of Philadelphia, delivered January 17, 1911.

The subject which I have selected to address you on tonight is one of the more recent additions to our knowledge of cardiac pathology. For many years a condition has been described in text-books of physiology as fibrillation, or vermicular or incoördinate movement, or sometimes as delirium; this delirium of the laboratory has been used in quite a different sense from the delirium cordis of the clinician, although curiously enough the clinical and physiological delirium prove to have an unsuspected relationship. This fibrillation is most readily induced by rapidly repeated electric shocks applied to the heart. When a rapid series of weak electric shocks is passed through a chamber of the heart, it responds with occasional extrasystoles, and if the strength of the shocks be increased an artificial rhythm may be elicited. As the strength of the current is further increased the rhythm accelerates, diastole becomes imperfect, and finally the chamber no longer reacts as a whole, but apparently individual fibres take up their own independent rhythm —fibrillation, incoördinate or vermicular contraction, or delirium. When this stage is reached, the chamber no longer expels its contents, and if the ventricle is the chamber involved, the circulation ceases and death results unless the normal rhythm returns. A number of poisons in large quantities have similar results—digitalis group, aconitine, barium, etc.—and it seems not unlikely that some cases of sudden death from "heart failure" are due to the ventricle passing into fibrillation. In dogs dying suddenly in the course of chloroform anesthesia, I have sometimes observed this fibrillation of the ventricle when the thorax was opened immediately.

When the auricle passes into fibrillation, the effects are not so serious, for the auricle is not essential to the circulation, and auricular fibrillation may last for hours in animals without serious consequences. When the electric stimulation of the auricle is of short duration, the chamber resumes its normal contraction immediately, but if the stimulation be continued for some time or if it be repeated, the fibrillation persists for some time after the electrodes have been removed, and after prolonged stimulation the phenomenon may continue for hours. Fibrillation of the auricle may be induced by the same poisons as that of the ventricle, and I have seen it occur in dogs from stimulation of the vagus. During fibrillation the auricle ceases to expel its blood and dilates widely. (Fig. 1.) The appearance of the chamber is very characteristic, but it is difficult to picture it unless it has been seen; instead of the short shock contraction which is characteristic of the normal auricular movement, one sees a quivering surface, each fiber of which seems to act independently of the others. A somewhat similar quivering is sometimes seen in the tongue and may be more familiar to you. Impulses continue to pass from the auricle to the ventricle as fast as they can be transmitted through the connecting band of His, but they are irregular, no two beats occurring at the same interval, and the strength of the contractions varying extremely. In particular there is no definite

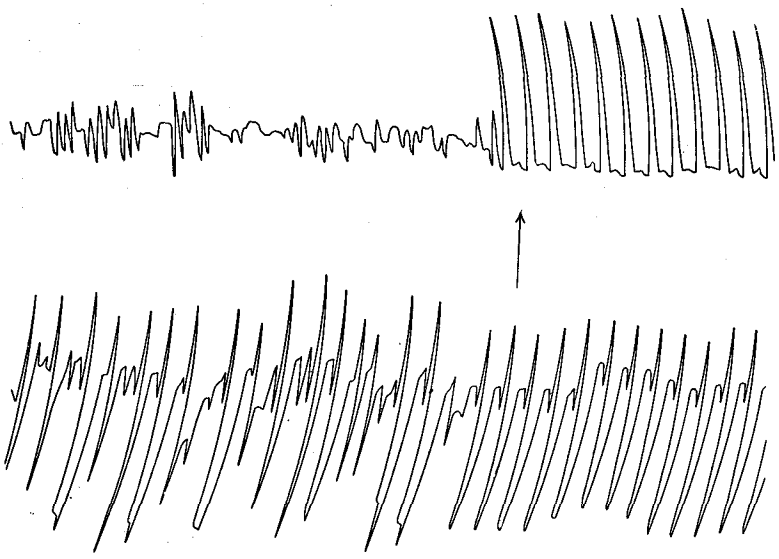

FIG. 1.—Tracing of the movements of the right ventricle (lower) and right auricle of the dog. To the right of the arrow, normal movements; to the left, auricular fibrillation from electrical stimulation of the auricle.

relation between the strength of the contraction and the preceding pause. These irregularities are, of course, reflected in the radial pulse and in the apex tracing. In 1899, I suggested that the tracing of the arterial pulse in the dog (Fig. 2) during auricular fibrillation presented a striking similarity to the pulse known clinically as delirium cordis, and two years later I had the opportunity of examining a case of rapid irregular rhythm in a patient, which appeared suddenly, lasted for an hour or two, and then changed to the normal rhythm for some hours (paroxysmal tachycardia). The sphygmographic tracing presented such a marked similarity to that obtained from dogs during auricular fibrillation that I concluded that in this patient the cause of the irregularity was the same—auricular fibrillation.

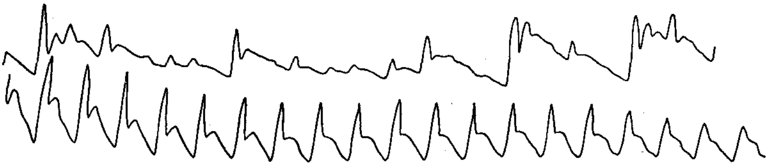

FIG. 2.—Tracings from carotid of dog. The lower represents the normal pulse; the upper was taken during fibrillation of the auricle from rapid electrical stimulation. (Cushny and Edmunds.)

Unfortunately the method of taking the jugular pulse was unknown to me at the time (1901) and there was no way of confirming my diagnosis. When I and Edmunds published this view in 1906,[2] it was received with sympathy but incredulity by several writers on irregularity of the heart, the difficulty raised by them being that it was impossible to conceive of auricular fibrillation persisting for weeks or even years, while cases of tachycardia were known to have lived for this time. When I put my view to Mackenzie, he was struck by its applicability to a series of cases which he had described in which every trace of auricular activity had disappeared, and in which he had concluded that the auricle was entirely paralyzed. He agreed that my view would equally well explain these cases, in which he had found hypertrophy of the auricle at autopsy, which scarcely agreed with a paralysis of this organ; but the idea of auricular fibrillation was new and unknown to him as to most clinicians, and though some mention of it may be found in his papers about 1907, he had abandoned fibrillation for another explanation. Quite recently our view had been proved correct by the researches of Rothberger and Winterberg,[3] and especially by those of Lewis. Auricular fibrillation is a common form of heart irregularity in man.

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[2] Paroxysmal Irregularity of the Heart and Auricular Fibrillation, AMER. JOUR. MED. SCI., January, 1906.

[3] Wien. klin. Woch., 1909, June 17, p. 839.

[4] Heart, 1910, i, 306.

in fact, is much the most common cause of serious and prolonged irregularity.

It is therefore of importance to consider the characters of this irregularity as seen in man. And these may be stated shortly: (1) Very irregular pulse, the strength of successive beats bearing no relation to the preceding pause, and (2) disappearance of all trace of auricular activity in the jugular, liver, apex, and esophageal tracings, and in the electrocardiogram. In mitral stenosis the systole of the auricle is indicated by a murmur (presystolic), but when fibrillation is present this also disappears. This condition of auricular inactivity was detected and first described by Mackenzie, to whom we owe the recognition of this form of heart disease as a distinct pathological entity; as far as the clinical picture is concerned, his work has not received nor needed any amplification from other hands. It has been known by many names, such as delirium cordis, nodal rhythm, ventricular form of the venous pulse, pulsus irregularis (perpetuus), but will, it is to be hoped, be known in the future by its essential feature, as auricular fibrillation.

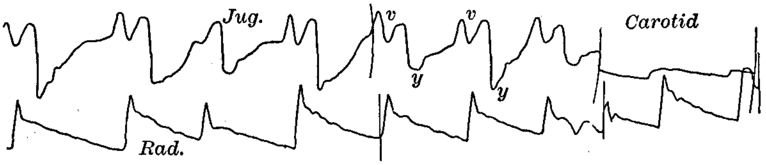

FIG. 3.—Radial pulse (lower) and jugular (upper) in a case of "ventricular form of venous pulse" (auricular fibrillation). (Mackenzie.)

The auricle ceases to expel its contents, and this is recorded in the jugular tracing by the absence of the *a* wave, and in long standing cases in which the auricle has lost its elasticity entirely, by the disappearance of the negative wave, which in the normal tracing marks the auricular diastole. The auricle remaining dilated throughout the cycle, the tracing of the vein must, in fact, be the same as if the jugular bulb was attached directly to the tricuspid valves. The tracing obtained from it, therefore, shows the negative wave when the blood flows onward into the ventricle, and as this chamber becomes filled, the tracing rises more or less slowly to be jerked up suddenly when the flow is arrested by the ventricular contraction, and to fall again when the way is opened again by the relaxation of the ventricle. In the normal pulse the same thing occurs, of course, the ventricular systole cutting off the inflow of blood into the ventricle. But here there is not stasis in the veins, for the blood flows into the auricle and the wave *v* is not very marked therefore. There may be superposed on the upper part of the curve several waves from the reflection of the carotid pulse and other causes, but the essential feature is the absence of *a* and its following negative wave.

Wenckebach and Mackenzie have observed occasionally in the venous tracing a series of undulations during the pause of inactivity of the ventricle. It is not yet determined whether these arise from the feeble movements of the auricle, but it seems not unlikely that these may be transmitted through the swollen vein when they are of the coarse variety and the beat is slow. Lewis has observed them also in the dog.

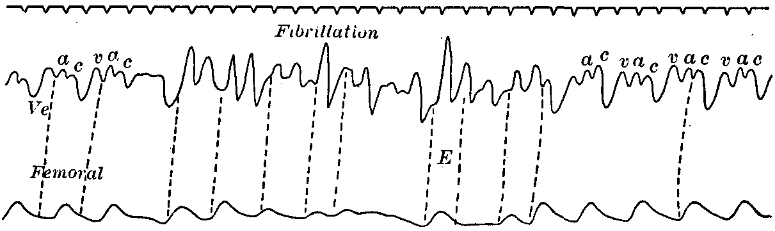

FIG. 4.—Jugular pulse (upper) and femoral (lower) in dog. To the left the heart is normal during three beats. Then auricular fibrillation is induced by electrical stimulation and again the normal rhythm returns in the last four beats. (Lewis.)

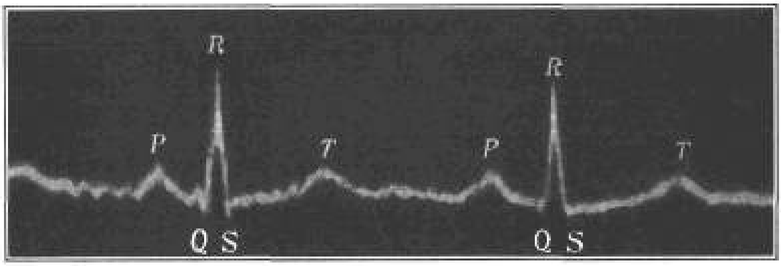

FIG. 5.—Normal electrocardiogram in human heart.

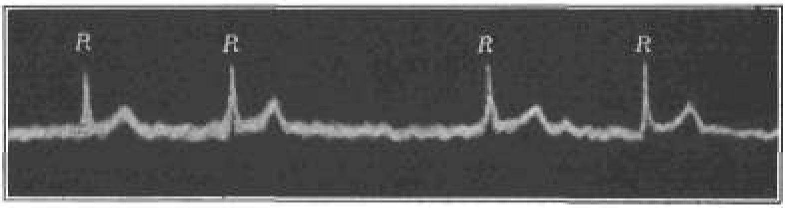

FIG. 6.—Electrocardiogram of dog during auricular fibrillation. (Lewis.)

It remains only to consider the changes seen in the electrocardiogram in this form of irregularity. When the electrical changes in the normal heart are recorded photographically by means of Einthoven's string galvanometer, each cycle is seen to cause a series of waves which have been lettered *P*, *Q*, *R*, *S*, *T*. Of these *P* arises from the auricle, and is positive, and is followed by the small negtive wave *Q*. Then *R*, a marked and sudden positive wave, followed by a depression, *S*, and a lower flat wave, *T*, all arise from the ventricular contraction. In auricular fibrillation in animals and in man, *P* disappears, the auricle not undergoing any active contraction. *R*, *S*, *T*, remain unaltered in essentials though, of course, the periods between each group vary in length, corresponding to the irregularity of the ventricular rhythm. The fact that *R*, *S*, *T* remain practically unaltered indicates that the ventricular contraction originates at the normal point and follows the normal course, and suggests that it is the response to an impulse descending from the auricle by the normal path through His' bundle. So far we have not progressed much further than with the jugular tracing which also shows the *a* to be absent. The convincing feature obtained by the electrocardiogram is the presence of a new series of undulations (450 to 900 per minute) on the curve, whether this is obtained from animals in fibrillation or from patients. These are seen throughout the electrocardiogram as far as they are not obscured by its other features and occur in no other known condition in either animals or man. They continue during the period of inactivity of the ventricle, and are more pronounced when the galvanometer is connected with the skin over the auricles than when the leads are taken from other parts of the heart. In fact, they may be little marked when the current is led off from the ventricles. And they are most marked of all when the fibrillating auricles of the dog are directly connected with the galvanometer. When the fibrillary contractions cease in experiments on the dog, these waves disappear, and in paroxysmal tachycardia in man they vanish also on the resumption of the normal

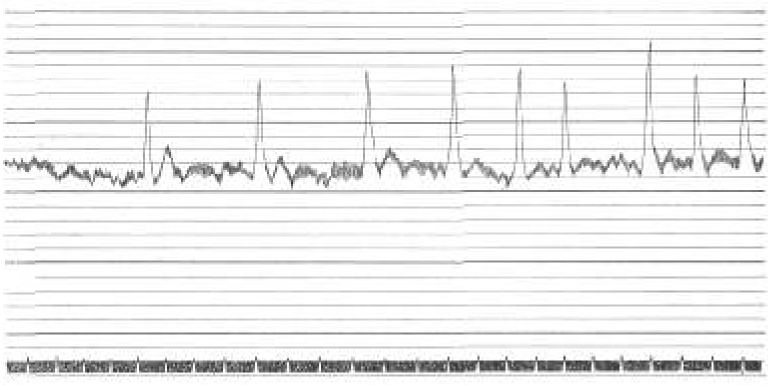
FIG. 7.—Electrocardiogram of patient during fibrillation of the auricle. (Rothberger and Winterberg.)

rhythm at the end of the paroxysm. I may be prejudiced in favor of a rather neglected offspring, but it seems to me that the observations of Rothberger and Winterberg and Lewis place auricular fibrillation upon an indisputable basis as occurring in man and as being the cause of the marked irregularity of the heart in question. But to those who are unfamiliar with the electrocardiograph, further and ocular proofs may be vouchsafed. It is true that no one has seen auricular fibrillation in a living man, though it occurs in the surviving human heart when it is perfused with Ringer's solution. But there is a disease in the horse marked by irregularity of the heart very similar to that met with in man and known as "tumultuous heart." This lasts for months and years in the horse, but renders it so weak and useless that as a general rule it is shot. In a horse destroyed in this way and immediately opened, Lewis found the auricle in fibrillation resembling that observed in dogs experimentally. The condition was of long standing, the proof is *ad oculos*, that the fibrillation is the cause.

During auricular fibrillation impulses are probably showered upon the auriculoventricular fibers in indefinite numbers, but these are capable of conveying only a certain proportion of them, and the rate of the ventricle is determined by the number it receives, that is, the number conducted by the bundle of His. As a general rule this is large, and the pulse is rapid (100 to 150). But in a certain proportion of cases the bundle is affected and can convey only a smaller proportion of impulses, and the pulse is then slow. The passage of the impulses is irregular and this accounts for the great irregularity of the ventricular beat and of the pulse. In one case Lewis[5] observed auricular fibrillation with complete block of His' bundle, so that the ventricle had its own slow rhythm, which was quite regular, while no sign of auricular beat could be found in the venous tracing; the case, in fact, was a typical one of heart block as far as the ventricle was concerned, and differed from the ordinary type only in the auricle fibrillating; frequent attacks of unconsciousness were observed in this case as in ordinary heart block in which the Stokes-Adams syndrome is presented.

Fibrillation of the auricle thus playing an important part in cardiac disease in man, it is of interest to note any further points known regarding it in animals, in which it has long been recognized. It may be caused by stimulation of the auricle with rapid electrical shocks, or by the use of certain drugs which act on the heart muscle, such as digitalis or barium in large doses. It is also induced occasionally by stimulation of the vagus in dogs, and stimulation of this nerve seems to favor its development generally, for Winterberg[6] found that weaker stimulation of the auricle caused fibrillation more readily during vagus stimulation than in other conditions. In a number of my earlier experiments on the heart I found the auricle in fibrillation and attributed this to reflex stimulation of the vagus. When fibrillation of the auricle has been developed, vagus stimulation in rare cases appears to arrest it, and in others renders the movement a finer one, but apart from this smaller movement it has no obvious result as far as the auricle is concerned. Vagus stimulation slows the ventricle, however, during auricular fibrillation, and renders the rhythm less irregular; this slowing of the ventricle arises from fewer impulses reaching it from the auricle from the passage through His' bundle being rendered more difficult through the inhibition. If the stimulation is strong in fact, complete heart block may be induced in some animals, and the ventricle then beats with a very slow and regular rhythm.

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[5] Quart. Jour. of Med., 1910, iii, 273.

[6] Pflüger's Archiv, cxvii, 223.

In man stimulation of the vagus by pressure on it in the neck has the same effect, the rate of the pulse slowing to a marked extent (Wenckebach)[3], but the fibrillation continues and the rapid rhythm returns when the stimulation ceases. Removal of the vagus influence by giving atropine quickens the pulse but does not remove the irregularity except in very rare cases, its only effect being to facilitate the descent of impulses through His' bundle. The accelerator nerves seem to have little effect on the fibrillation, but may retard it to some extent (Winterberg).

The etiology of the condition in man is not yet determined, in fact its existence has been definitely proved only within the last year. In certain cases, as for example, that first described by Edmunds and myself, the attacks of fibrillation come on and relapse to ordinary rhythm so suddenly that one is disposed to regard them as due to some nervous influence, and since in animals vagus stimulation favors fibrillation and may induce it, I think it is justifiable to regard some of these cases as of nervous origin, as, indeed, we did in our first case. I am encouraged to this view because Hering found in one case that atropine, by removing the vagus action, restored the regular rhythm. Rothberger and Winterberg suggest that in early cases in which the fibrillation is not fully established the action of the vagus may be essential to its maintenance, and the removal of this favoring condition by atropine may be sufficient to restore the normal rhythm. When the fibrillation is fully developed, however, atropine does not arrest it. In other cases fibrillation occurs after the presence of more or less numerous auricular extrasystoles have been observed for some time previously; these extrasystoles indicate an abnormal irritability of the auricle muscle and the fibrillation is merely a further development of this condition. In animals, stimulation of the auricle with weak shocks causes extrasystoles and under stronger stimulation fibrillation is developed. In some cases both these factors, inhibition and abnormal irritability,

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[7] Brit. Med. Jour., November 10, 1910, p. 1604; Fig. 17.

may contribute to the result. Thus in two cases which I have seen there was an extremely rapid regular pulse running to over 200 per minute, the rapidity being due apparently to extreme irritability of some point in the auricle which had taken up the rhythmogenic function. In both these cases digitalis slowed the pulse and at the same time induced short phases of fibrillation; I suggest that here the two factors of excessive irritability of the auricle and inhibition (from digitalis) were present and the fibrillation resulted. In a third case, in which the pulse was regular and rapid (90 to 100), digitalis in large doses induced sudden auricular fibrillation with a slow pulse (60 to 70), which after some hours as suddenly assumed its former rate and rhythm. Here I cannot help thinking that the arrhythmia was induced by the inhibitory action acting on a very excitable auricle, the excitability perhaps being due to the muscular action of digitalis.

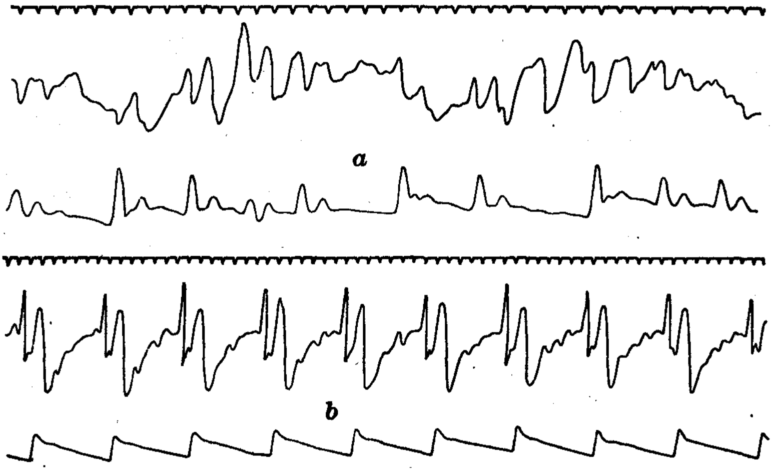

FIG. 8.—Radial and jugular tracings in patient during auricular fibrillation; (*a*) before treatment, (*b*) after treatment with digitalis.

Fibrillation may be suddenly developed and pass off again in a few minutes or a few hours or days; it is, in fact, present in many cases of paroxysmal tachycardia, though perhaps not in all. On the other hand, it may persist for many years, in some cases not interfering with ordinary avocations, but generally reducing the patient to a more or less invalid existence. In some cases sudden death has occurred probably from the fibrillation extending to the ventricle.

The diagnosis may be made with a fair amount of accuracy from the radial pulse alone. No condition is known in which the same degree of irregularity and the same variation in the strength of the contractions with respect to the interval preceding them are met with. On the other hand, in young persons, fibrillation may be present without any striking irregularity of the pulse. But an absolutely certain diagnosis requires the use of the jugular tracing. When there is no *a* wave in this and the radial pulse has the characteristic irregularity the condition is certainly auricular fibrillation.

As regards the treatment of the condition, no disease of the heart responds so satisfactorily to digitalis and its allies as auricular fibrillation. In fact, were it not for its almost specific action in fibrillation it may be questioned whether this series would enjoy the reputation it has in heart disease. But not all cases of fibrillation react thus brilliantly to digitalis; the slow irregular heart in which fibrillation is accompanied by impaired conduction is comparatively little improved. The condition which responds to digitalis most readily and satisfactorily is the rapid irregular heart of old rheumatic disease, in which fibrillation is present along with good conductivity through the bundle of His and a readily responding ventricle. Here digitalis reduces the pulse rate rapidly from 120 to 150 to 60 to 70, and as the pulse falls the whole of the symptoms improve. If the drug be pushed the pulse continues to fall and may reach 40 to 50. Beyond this digitalis generally induces other symptoms from the gastro-intestinal tract so that its dose has to be reduced. If it is abandoned altogether the previous condition soon returns, often in a less marked degree, and we have found it advisable to continue the treatment for many months. Digitalis does not arrest the fibrillation of the auricle, but it certainly reduces the number of impulses which reach the ventricle and thus lessens the rate of that chamber. I am not convinced that this reduction of rate, which is undoubtedly mainly inhibitory in nature, is the only effect of digitalis, though it is the only one which we can ascertain with certainty at present, and undoubtedly plays an important role in the action.

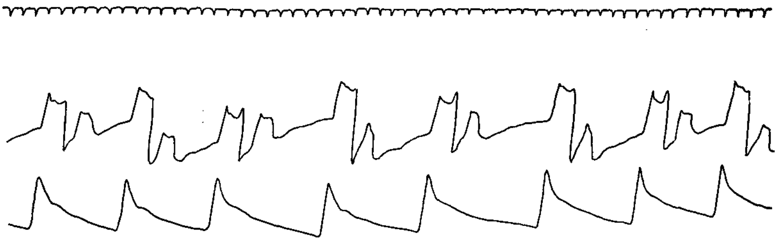

FIG. 9.—Radial (lower) and apex (upper) tracing in fibrillation treated with large doses of digitalis. Coupled beats (begeminus) are seen in the apex tracing, but the extrasystoles are too weak to cause a pulse in the radial.

When digitalis is administered in auricular fibrillation and the pulse is slowed, the rhythm becomes more regular and the beats more equal in strength, though complete regularity is not attained unless complete block is induced. If the drug is pushed a curious new form of irregularity often presents itself in coupled beats or continuous bigeminus in which each beat of the ventricle that arises in the normal way from an auricular impulse is followed by a contraction arising from the ventricle itself.

In connection with the treatment of fibrillation with digitalis, I may again remind you that an attack of this condition may in certain circumstances be precipitated by this drug.

In animal experiments various other measures have been found useful in arresting fibrillation, but these are all inapplicable in human patients. Thus I have found that cooling the heart with iced saline is often successful, while Gottlieb found that camphor perfused through the vessels seemed to have some effect in retarding its development. Hering recommends the perfusion of potassium chloride until the heart ceases all movement, and then restores it by means of Ringer's solution perfused through the coronaries. I merely mention these methods to show their inapplicability, so that no one may be induced to attempt the treatment of this condition by means of potassium salts.

PERFORATED ULCER OF THE STOMACH AND DUODENUM.

By George G. Ross, M.D.,

SURGEON TO THE GERMANTOWN HOSPITAL; ASSISTANT SURGEON TO THE GERMAN HOSPITAL, PHILADELPHIA.

Within the past two years I have had four cases of perforating ulcer of the stomach or duodenum, several of which show points of marked interest in reference to the diagnosis, treatment, and end results. They are as follows:

Case I.—Mr. Samuel M., referred by Drs. Ullom and Johnson, was admitted to the Germantown Hospital September 17, 1908. He gave a history of chronic dyspepsia, associated with vomiting and localized pain in epigastrium. He had been a sufferer from these symptoms for years. At 11 A.M. on the morning of admission he was seized with sudden, agonizing pain in the upper abdomen. The pain, which was extremely severe, radiated to the back and seemed to have its point of greatest intensity in the right iliac fossa. His abdomen was generally rigid, being most marked in the epigastric region. He was exquisitely tender over McBurney's point. There was no very great tenderness of the upper abdomen. He was operated on at 1 P.M., two hours after the onset of the attack. When the peritoneum was opened, gas and gastric contents escaped. The greatest quantity of fluid and debris was found in the right iliac fossa, although there were large quantities